among well-to-do people, but is the average for the mass of city dwellers in this locality. Bronchopneumonia is preëminently a disease of the first two years of life, and after the third year is relatively uncommon. Lobar pneumonia is the type of the disease which is present after the third year in practically all cases of pneumonia, if those cases which are frankly secondary to some other condition, such as one of the infectious diseases, or where the pneumonia occurs as a terminal infection, are omitted. Lobar pneumonia *per se* is a common condition in the first and second years of life, being much more frequent than is commonly supposed. The infection which is the etiological factor of lobar pneumonia is always the pneumococcus, while a bronchopneumonia may be due to a number of organisms, such as the streptococcus or the influenza bacillus, occurring alone or as a mixed infection. If pneumococci are present in bronchopneumonia they are usually one of a group of various organisms, or at least are of a low virulence, and resemble the organisms commonly found in the mouth. The pneumococcus may be divided into four general groups, each being made up of many races which are closely related. By the method outlined a pure culture of pneumococcus may be obtained and the group determination completed in about twenty-four hours from any given case of pneumonia. The division of the pneumococcus into groups is of the greatest importance from the stand-point of treatment. Obviously the treatment of a lobar pneumonia due to a pneumococcus of Group II with a serum or vaccine prepared from an organism of Group I is a waste of energy and might be positively harmful in its effect.

Pneumonia is the most common, is the most fatal, and is the least studied disease that occurs among children. Any work which brings new facts relating to its treatment or new possibilities of improved methods and prevention is worthy of the careful consideration of all those who are interested in the welfare of children. It is hoped that the division of pneumonia into groups will eventually bring about the treatment by specific sera or vaccines, replacing the symptomatic treatment of today.

ACUTE CEREBELLAR ATAXIA IN CHILDREN: REPORT OF A CASE, WITH RAPID AND COMPLETE RECOVERY.

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THE condition about to be described appears to be an uncommon one. On this account it seems well to put the following case on record and to review the already published cases of a similar nature.

Susie R., aged five years. The family history showed nothing of importance. There were three brothers and one sister, who were well. The patient had always been a healthy child, except for occasional attacks of bronchitis. There had been no illness of any sort in the household recently. On Monday, January 4, she suffered from cold in the head and a bronchial cough. An epidemic of influenza had been prevailing in the city. The symptoms continued upon Tuesday, January 5, although the child was running around as usual. On Wednesday, January 6, she was worse, and Dr. Wm. McCombs attended her for the next several days, and put her to bed. She had a heavily coated tongue, headache, and a temperature from 98° to 102°. She continued about the same from January 7 to January 9, when her temperature had become normal. By Sunday, January 10, she was apparently quite convalescent, and was allowed to get out of bed. This day she was cheerful, playing, and seemed entirely well. In the evening, however, she complained of slight earache, from which she had never suffered before, but on Monday, January 11, she was up and about. Toward evening she began to complain that her hip hurt her, and she was hardly able to walk. On Tuesday, January 12, she vomited before she was out of bed. Later in the course of the morning she got up, but in the afternoon vomited several times and went back to bed. She still complained of her hip hurting, and any pressure upon it seemed to make her shake all over. The mother noticed also that there was a jerking, irregular character to her speech. These were the first distinct nervous symptoms, and her mind seemed entirely clear. On the morning of Wednesday, January 13, the child was scarcely able to stand, and the mother now noticed the irregular movements of the eyes and eyelids. On this day she seemed dazed. Dr. McCombs described it as "a state of fear." She could not well be quieted, and in the afternoon her mental state became worse, and in addition she could not sit unsupported. About 9 o'clock in the evening she began to scream violently, sometimes for an hour at a time, almost without pause. This continued until 4 o'clock on the morning of the next day, January 14. She was pale and trembling; half delirious; there was apparently no disturbance of the tactile sensation, and she could feel wherever she was touched. After about two hours' sleep she started to scream again, and was taken during the day to the Children's Hospital, four days after the onset of the acute symptoms.

Examination on admission, January 14, showed a well-nourished child of good color. The mind did not appear entirely clear, but there seemed to be no special excitability. She could utter a few words, but with great difficulty, and the speech was very jerking and irregular. There was a very high degree of lateral nystagmus nearly always present, but growing worse in paroxysms and increased on ocular exertion, and accompanied by a fluttering movement of the eyelids. The patellar reflexes were increased. There was a decided incoördination of the muscular movements of the extremities. A few mucous rales were heard in the lungs; respiration was regular. The heart showed nothing abnormal.

I made an examination of the child for the first time upon the next day, January 15, with the following notes: "The mental condition must certainly have improved, for it now seems nearly normal. She answers questions clearly, but after some delay and with a slight trembling of the voice. There is a curious, very marked fluttering of the eyelids which is not constant; and with this a rapid nystagmus, both lateral and vertical, lasting for a moment and then for a moment disappearing. No paralysis of the ocular movements can be found, and no facial paralysis. The child can whistle and show her teeth when directed. Her grip is good. There is trembling and a very decided ataxia in the movements of the arms and of the hands on attempted effort, apparently not increased by closing her eyes. The patellar reflexes are active, and there is slight ankle-clonus of the right foot, and a suspicion of this in the left foot. The Babinski symptom is absent. She can stand only when supported, and with her feet broadly separated from each other."

On January 16 and 18 Dr. Isaac Jones made careful examinations of the ears to determine whether the nystagmus was otitic in origin. The summary of his report reads as follows: Typical nystagmus and unbalancing of the control of the eye-muscles are present. This is not due to any lesion of the ear, or any disturbance of the vestibular apparatus, because (1) spontaneous nystagmus on looking to the extreme right or left is decreased instead of increased; (2) the hearing is normal; (3) stimulation of the semicircular canals gives the correct normal nystagmus, controlling the oscillations and converting them into rhythmic nystagmus of a vestibular character, with a slow and rapid component. The nystagmus-circuit is, therefore, intact on both sides; indicating normal semicircular canals, eighth nerves, region of Deiters' nucleus, the posterior longitudinal bundle in the pons and its connection with the third and fourth nuclei, and, finally, the third and fourth nerves themselves.

On January 19 an ophthalmological examination was made by Dr. H. M. Langdon and the following report rendered: "Palpebral fissures about equal. In the right eye the pupil measures 4.5 mm.; left eye, pupil slightly smaller. On admission of light the accommodation is normal in each eye. The ocular rotation seems full and equal in all directions, the visual axes being parallel. The eyes are steady on fixation, but on rotation in any direction there develop a few quick nystagmic jerks, usually about five or six oscillations in a group, and then steady fixation will be resumed. The movements were usually accompanied by curious fluttering of the eyelids. The disks are slightly full, normal in color, with clear margins. There are no changes of the fundus."

Improvement went on with great rapidity. By January 23, nine days after admission, the child could sit up in bed unsupported, with some ataxic movements of the arms on attempting to balance herself. While standing on the floor there were some oscillatory movements of the entire body, but she could now walk if supported by one arm, and her gait was normal, except for a degree of weakness and uncertainty. On closing her eyes she was unsteady, but not to a degree constituting Romberg's symptom. The grip was only fair, but equal on both sides. The patellar reflexes were slightly increased on the left side. No ankle-clonus or Babinski reflex was present. The eyes exhibited a rotary nystagmus as observed before, but to a much less degree. The sensorium was entirely normal; the child answering questions and conversing freely, in contradistinction to the condition earlier in her history, when, although she would answer questions, she made no effort to speak at other times. The tongue was protruded with ease in any direction. No facialis symptom could be elicited. She complained of her feet being constantly cold. She could feed herself fairly well and touch her nose with the eyes closed.

By February 1, seventeen days after admission, it was noted that she still walked with some unsteadiness, but without any support, and with the feet rather wide apart and the body bent somewhat backward in the effort to balance herself. The nystagmus had disappeared except on continued ocular exertion to the extreme of the ocular field. On February 10, one month after the onset of the acute symptoms, she was discharged from the hospital, and the following note was made: "Since admission there has been an entire change of mentality from sluggishness, haziness, and dulness to a condition of alertness and brightness."

The temperature while in the hospital was afebrile, except for a rise to 100° on a single occasion. The Wassermann reaction taken on January 18 was negative and the von Pirquet faintly positive. Lumbar puncture on January 21 gave a dry tap. The urine was examined on a number of occasions, and was practically always negative. On one examination a faint trace of albumin, cylindroids, an occasional hyaline cast, and a few leukocytes were reported. Examination of the blood on the day of admission showed a leukocyte-count of 11,600. The differential count gave polymorphonuclears, 57 per cent.; lymphocytes, 31 per cent.; mononuclears, 7.5 per cent.; transitionals, 3 per cent.; eosinophiles, 1.5 per cent; basophiles, 0.

Treatment consisted on admission in the administration of a purgative and of sodium bromide. After this, on the ground that the condition might be toxic, bicarbonate of soda was given every four hours and enteroclysis every six hours. This treatment was continued until recovery was well advanced.

The noteworthy features in this case consist in the rapid development of symptoms without discoverable cause, unless possibly the child had suffered from influenza; the very uncommon degree of nystagmus; the ataxia of the extremities; disturbance of sensorium; affection of speech; slight increase of reflexes, and the rapid recovery, complete in one month from the onset.

We have here symptoms which, on the whole, point chiefly to some disorder of the cerebellum. That this was the only portion of the brain affected would seem questionable. As Batten[1] has pointed out in an able paper upon "Ataxia in Childhood," it is impossible to draw a sharp line between the cerebellar and the cerebral cases. In some the symptoms pointing to cerebral involvement predominate, and in others, as in the case reported, the cerebellar symptoms are in the ascendency.

In the case I have described, such systemic disorders as Friedreich's ataxia and the cerebellar ataxia of Marie were naturally out of the question. Tumor and abscess of the cerebellum, at first thought to be possibilities, were excluded by the course of the disease and by the examination of the eye-grounds; and that the nystagmus was not otitic in origin was shown by the application of Bárány's tests, although this was not certain proof that the symptom depended upon a cerebellar lesion.

Fickler[2] gives a convenient classification of the diseases of the cerebellum, which may profitably be considered in the effort to place the case described. The first group is that of congenital cerebellar ataxia, from which this case is naturally excluded by the age. The second consists of the acquired cases, which are subdivided into the chronic progressive and the acute forms. The latter, to which this patient naturally belongs, is again divided into (*a*) traumatic (*b*) encephalitic, and (*c*) toxic. According to this classification the encephalitic cases follow especially the infectious diseases. The toxic cases would be represented in early life by those depending upon gastro-intestinal auto-intoxication. The anatomical difference would appear to be that in encephalitis there is an actual inflammatory process active, and in the toxic cases the condition in the acute stage is one of cellular degeneration. Clinically it would seem difficult to make a sharp distinction, and the two may be conveniently grouped in this connection under the heading of acute non-suppurative encephalitis, or acute hemorrhagic encephalitis.

The acute infectious diseases are the most frequent of the causes mentioned. In epidemics of poliomyelitis, too, cases are not infrequently seen with the symptoms of encephalitis, alone or in combination with spinal symptoms. The process in this disease is known to be an inflammatory one; and yet the recovery may be so complete in some instances that, in the absence of any other satisfactory explanation, the case recorded here might perhaps be assigned to this group; in this instance, most unusually, the cerebellum bearing the brunt of the attack. Against this supposition, yet not completely excluding poliomyelitis as the etiological factor, is the fact that nearly always the lesions in this condition are more widely disseminated than in the present case.

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[1] Brain, 1905, xxviii, 484.

[2] Deutsch. Zeitschr. f. Nervenheilk., 1911, xli, 306.

In the large majority of cases of uncombined acute hemorrhagic encephalitis the process is limited to the motor region of the cerebrum. In many it is seen also in, and in a very few cases it is confined entirely to, the region of the pons and medulla. In other cases, much less numerous than the first class, the encephalitis, as stated, attacks especially the cerebellum. In this event there is the comparatively sudden development of the symptoms of encephalitis in general, such as fever, unconsciousness, convulsions, and the like, lasting a variable time, although in the end the cerebellar symptoms predominate. The most characteristic of these are ataxia, staggering gait, and vertigo; although often associated, but not certainly cerebellar in origin, are nystagmus, disturbances of speech, and tremor.

The prognosis of the great majority of cases of acute non-suppurative encephalitis, whatever the location, is favorable as far as life is concerned, and favorable, too, as regards improvement; but less so as regards complete recovery of power and of a normal mental condition.

Batten details several cases similar in many respects to the one I have reported, and publishes others collected from medical literature; and there are still a number of others not found in his series. I append abstracts of all the cases I have been able to discover. Only those are included in which the course of the disorder was sooner or later arrested and improvement followed. Those showing a progressive increase in the symptoms do not belong to this category.

(1) Shepherd (*Med. Times and Gazette,* 1868, i, 144). Girl, aged five years. On the second day of scarlet fever she became speechless. Examination a month later showed her unable to speak, write, or stand; her head and arms fell forward; her legs were weak. After three weeks she improved rapidly and could walk, although with an ataxic gait. Gradually she became more intelligent. In about three and one-half months from the onset she could play about the ward, although her movements and speech were far from normal.

(2) Schepers (*Berl. klin. Wchnschr.*, 1872, ix, 517). Girl, aged eight years. On the fourth day of measles she became unconscious, and later exhibited aphasia and then marked ataxia of the limbs. The intelligence seemed unaffected; the sensibility normal. In one and one-half months she began to speak, but with an altered

voice; she could stand supported, but not walk; there was great ataxia in the hands. A week later she walked with an ataxic gait. Memory had been largely lost, but she learned rapidly, and several months later *rejoined her original class in school.* Schepers speaks of the favorable outcome, but does not state whether any traces of the disorder remained.

(3) Feith (*Allg. Zeitschr. f. Psychiat.*, 1873–4, xxx, 236). Boy, aged five years. *Typhoid fever was followed by* aphasia, ataxia, and unconsciousness. After three weeks improvement took place in the mental state, but he still could not stand. Later he had psychic disturbances and a distinctly ataxic gait, and this condition lasted three weeks. About two months after the onset he seemed to have recovered completely. No reference is made to any ataxia of the upper extremities.

(4) This case is the only one in the series following trauma. The question could be raised whether it should properly be included here, inasmuch as the ataxia was of slower development than in the other instances. It seems to me, however, rapid enough to be called acute:—

Bastian (*Lancet,* 1878, ii, 207). Girl, aged ten years. She fell down stairs striking the occiput; experienced no loss of consciousness, but suffered from pain in the head on the next day. Two weeks later she had another fall on the occiput. After this she grew worse, and in two weeks was having vomiting and occipital pain. She was sent to the hospital eight weeks after the first fall. One week later, when attempting to get out of bed, she was found to stagger badly. Soon after this the sight became impaired. When seen again six months after the first accident she had a drunken gait; incoördination of the hands; optic neuritis; pain in the head; normal intelligence. She grew worse for a time, but after about three months improvement began, and in two months more she had perfect power in the upper extremities. By about a year after the fall she was entirely well, except a slight affection of gait.

(5) Lenhartz (*Berl. klin. Wchnschr.*, 1883, xx, 312). Boy, aged eight years. Acute dysentery was followed promptly by a brief maniacal state, and then entire loss of speech; anesthesia; paralysis of the sphincters, and mental weakness. Two weeks later he had nystagmus and marked ataxia of the head, extremities, and trunk. The aphasia and ataxia lasted for months, and he could not speak for three months. At the end of two and a half years there was only a little ataxia and slowness of speech and a deficiency in intelligence.

(6) Hammarberg (*Nord. med. Arkiv.*, 1890, xxii, No. 23). Male, aged seven years when he had pertussis, and soon after this "inflammation of the brain," with unconsciousness for a short time. This was followed by rhythmic swinging movements of the head; incoordination of the arms; loss of speech; inability to walk, and mental

disturbance for a short time. The condition remained unchanged for three months and then gradually improved. About one year after the onset the ataxia had lessened and he could walk unaided, but the gait was uncertain and reeling. The patient finally returned to school, but his gait continued to be always uncertain; the speech scanning and slow; he could not write well, and his disposition was altered for the worse. At the age of twenty-two years he had inflammation of the lungs, and after this gradually became insane and died at the age of twenty-four years.

(7–9) Lüthje (*Deutsch. Zeitschr. f. Nervenheilk.*, 1902, xxii, 280). Three cases occurring in one family, all developing during severe typhoid fever.

(7) Boy, aged ten years. Unconsciousness developed on the fifth day of the fever. This persisted for seven weeks, and afterward he remained dazed for over a month more and could not speak. During this time there were rhythmic movements of the head. When discharged from the hospital, four months after the onset, he could walk only with support; had ataxic movements of the legs and of the left arm; disturbance of speech, and ataxic nystagmus.

(8) Girl, aged seven years. Had unconsciousness for weeks, with oscillatory movements of the head, grinding of the teeth, and twitching of the face. She gradually improved and then showed decided ataxic movements of the extremities. Two and one-half months after the onset there were still imperfect speech; marked ataxia of extremities; unsteady gait; active tendon reflexes, and ankle-clonus. The sensibility was not affected, and there was no nystagmus or psychic disturbance. Three weeks later the gait and speech were still affected, but the upper extremities were normal.

(9) Boy, aged six years. He had a dazed condition for one and one-half months, and did not speak for two weeks more. Meantime there were rhythmic movements of the head, and marked ataxia of the trunk and extremities. No effort to speak was made until after one and one-half months, and then the speech was slow and scanning. He improved rapidly, and about two months from the onset he had remaining only a slight disturbance of speech and of gait.

Lüthje summarizes the principal symptoms of all three cases as follows: Severe typhoid fever followed by unconsciousness lasting for weeks; gradual recovery of mentality; severe ataxia in all the muscle groups, without paralysis or disturbance of sensation; slight disturbance of intelligence affecting the memory; scanning speech and increased reflexes.

(10) Taylor (*Lancet*, 1904, ii, 1416). Boy, aged four years. Six weeks before he was admitted to the hospital he had a slight fall, but without striking the head; three weeks later he had pertussis; four days before admission he developed twitching of eyes,

hands, and feet, and was unable to walk. Examination showed trembling of the limbs and the trunk, suggesting disseminated *sclerosis; inability to speak plainly* or to walk alone; nystagmus. In twenty-two months he could walk with a drunken gait when supported; the arms were ataxic; the speech hesitating; the *mental condition good.* By *three and one-half* years after the onset he was reported to be nearly well, although the speech was still a little imperfect. Examination over twenty years later showed the recovery complete.

(11) Voelcker (*Brain*, 1905, xxviii, 360). Girl, aged four and a half years. Scarlet fever was followed by unconsciousness lasting two weeks; no *vomiting.* *Then* the *speech* was found to be lost and she could not feed herself. Improvement gradually followed and when examined three months later she could not stand; the gait was ataxic when supported; there was some ataxia of the upper extremities; tremor of the head; brisk knee-jerks; variable plantar reflexes; no ankle-clonus; slow and indistinct speech. Two months later (five months after the onset) she showed little improvement; the knee-jerks were less active; the ataxia less; the mental condition improving. One and a half months after this the symptoms were still very marked.

(12) Guthrie (*Brain*, 1905, xxviii, 363). Girl, aged seven years. During measles there occurred vomiting, convulsions, squint, and unconsciousness. After five days she was better, but *could not* speak. Aphasia persisted, and there developed exaggerated knee-jerks; incoördination of upper and lower extremities; anesthesia and loss of sphincter-control. She improved *gradually, and in three months* could stand, talk, and feed herself. Sixteen months after the onset there were drawling speech; slight ataxia; tremor of the upper extremities; ataxic gait; brisk knee-jerks; *ankle-clonus at times.* She was mentally slow.

(13) Batten. First case. (*Brain*, 1905, xxviii, 489.) Girl, aged four and a half years. Immediately after *influenza* she *developed vomiting, and* in a week paresis of the legs and affection of speech; slight involvement of the sphincters; no loss of consciousness and never headache. A month and a half later she showed a wildly *ataxic gait when supported*, and could barely stand alone. There were marked incoördination of the hands; somewhat bulbar articulation; slight paresis of the left side of the face; active knee-jerks; *doubtful ankle-clonus.* There was no nystagmus. Examination three months after this showed that recovery was complete.

(14) Batten. Second case. (*Ibid*,, 490). Boy, aged three and a half years. *Measles* was *followed* by convulsions and unconsciousness lasting a week. Two months later there were slow speech; marked ataxia of the arms and legs; slight ankle-clonus, and perverted moral sense.. There was no nystagmus. After one and a half years he was very much better. When seen last, April, 1907

(*Clin. Soc. Trans.*, 1907, xi, 276), there was still slight incoördination of the legs, although the gait was good; the speech was rather slow and hesitating, and the mental condition rather backward for the age.

(15) Batten. Third case. (*Clin. Soc. Trans.*, 1907, xi, 276). Boy, aged eleven years. Without known cause he developed headache and on the next day falling; inability to feed himself, and vomiting. On the sixth day he had a drunken gait; ataxia of both legs, but less in the hands; nystagmus; active knee-jerks; loss of tone in the limbs; no affection of sensation. He improved rapidly and in a little over three weeks showed only slight ataxia of the lower extremities.

(16) Batten. Fourth case. (*Ibid.*, 277). Boy, aged twelve years. He had had petit mal for some years. After a severe attack he suffered from unconsciousness for three days. Following this there were no control over the arms or legs; slow and jerking speech; unsteadiness of the trunk and head; poor mental condition; intention-tremor; no vomiting or headache. He had not been able to walk for over six months, and when aided had a wildly ataxic gait. The knee-jerks were active. He improved but little while under observation.

(17) Nonne (*Neurolog. Centralbl.*, 1909, xxviii, 885). Boy, aged twelve years. During an epidemic of poliomyelitis he developed an encephalitis with cerebral symptoms, especially Jacksonian epilepsy, and subsequent hemiplegia. He was unconscious for a week. After subsidance of the cerebral symptoms, he showed general disturbance of coördination, affecting even speech and the muscles of respiration. In the course of two months the symptoms subsided, and in three months he was completely well.

Among more than doubtful cases not included in this series may be mentioned the following:

Marie and Joltrain (*Rev. Neurolog.*, 1910, xx, 123). Boy, aged fourteen years. He had typhoid fever at five years, followed by difficulty in speech, in walking, and in some of the movements of the extremities. The condition steadily grew worse. There was marked ataxia with complete abolition of reflexes. The mental state was normal.

This case, although beginning apparently acutely, had shown such a progressive increase of symptoms that it is rather to be placed in the category of "chronic progressive ataxia."

Baudoin and Français (*Rev. Neurolog.*, 1910, xx, 139). Girl, aged seven and a half years. Epileptiform seizures began at three months and continued with irregular and increasing frequency. When examined by the writers at seven and a half years of age the intelligence was little developed; she could express herself very imperfectly; there were slight increase of the knee-jerks on the left side; some hesitation and trembling in the upper and lower extremities, and a cerebellar gait. There was no nystagmus.

The doubt in this case is principally as to the acuteness of the origin, which may, in fact, have been congenital.

Williamson (*Proc. Royal Soc. of Med.*, 1912–13, Section on Diseases of Children, 41). Boy, aged ten years. Tremor began three and a half years before, and the child lost strength and grew less bright. Examination when brought to the hospital showed tremor in the arms and tongue; ataxic gait; brisk knee-jerks; no nystagmus. Seven months later there had been no marked progression of symptoms. A doubtful slight lateral nystagmus was present.

Williamson says that juvenile paralysis could be thought of in the diagnosis. There is nothing in his history indicating an acute onset, and the case hardly belongs here.

The following analysis and conclusions may be formulated based upon the 18 cases, including my own. They are necessarily incomplete, owing to the absence of sufficiently full details in some instances.

The immediate apparent causes of the attacks in the cases as reported are divided into scarlet fever, 2 cases; measles, 3; typhoid fever, 4; pertussis, 2; influenza, 1; poliomyelitis (?), 1; epileptiform convulsions, 1; trauma, 1; dysentery, 1; not discovered, 2. Possibly my own case, placed in the last class, could with reason be assigned to influenza. The preponderance of acute infectious diseases is very evident. Only 1 case followed trauma. It does not appear that the pertussis reported in 2 instances was of sufficient severity at the time to put these cases in the class of traumatism from violence of coughing; but possibly the case following epileptiform convulsions could be placed here. The instance of an attack following dysentery might possibly be an example of ataxia of toxic origin, but the duration was almost too long to permit of this conclusion. Ten of the patients were boys and 8 girls. The age at the time of onset ranged from three and a half years to twelve years, 10 of the patients being six or more years of age.

That the condition present is in fact dependent upon a lesion of the cerebellum is, in a way, an assumption. Only in the patient seen by Hammarberg did the case come to autopsy, and here very serious alterations of the cerebellum were found. This, however, was years after the first appearance of symptoms, the patient meanwhile having become insane and developing new somatic disturbances; and to how great a degree the early acute manifestations depended upon the lesions found must remain uncertain. The complex of symptoms based upon the composite of all the cases seems sufficient, however, to warrant a belief in the cerebellar origin. That the malady is not dependent upon vestibular or labyrinthine disease was shown in the case now reported by the application of Bárány's tests, and in none of the patients was any impairment of hearing noted. Vertigo, so characteristic a cerebellar or labyrinthine symptom, is not referred to in any of the reports.

The early age of most of the patients makes the identification of this symptom a matter of difficulty.

Disturbances of the sensorium were present in the early states of a large number of cases. Unconsciousness is mentioned in 11 instances, sometimes of brief duration, sometimes lasting for weeks. Convulsions were occasionally seen; delirium was also observed. All these may be classed among the symptoms common to any severe intracranial lesion, or they might be the evidence of a complicating disturbance in other regions than the cerebellum. Some affection of mentality, apart from unconsciousness, was present in 12 instances. In most of these it was of brief duration, but in a few it persisted in some form for a longer time. In such it, of course, indicated lesions elsewhere than in the cerebellum alone. Regarding the disturbance of speech, it is interesting to observe that in 8 cases the patient was for a time entirely unable to speak, and in some instances this condition lasted for months. Later, after the power to speak had been regained, in all but 3 of the cases some affection of it remained for a variable time, and in many was still present when the patient was last seen. It is described as "slow," "drawling," "scanning," "jerking," or "irregular." The affection of speech might with propriety be considered an extracerebellar disturbance, but certainly in some cases at least, and perhaps in most of them, seems not so much to depend upon an involvement of the centres for speech as upon an inability to articulate properly—an ataxic condition. It is possible that in producing the speech-disturbance the medulla may also be involved, and the same is true of the vomiting, which was seen early in some of the cases.

The tendon reflexes were found "brisk" or "increased" in 9 instances. In the others nothing is said upon the subject. Ankle-clonus was present in 4 cases. It is evident, then, that there is a distinct tendency to increase of the reflexes in this disorder, pointing toward the cerebellar involvement. In a few cases "weakness" or "hypotonia" of the limbs is mentioned, but in others it is distinctly stated that no weakness or paralysis was discoverable. In general it is a fair conclusion that the inability to walk or to use the arms depended upon the ataxia rather than upon paresis. The ataxia was noted in every instance; in the legs in all, in the arms it appears to have been present in all but one case. Not infrequently the trunk and the head shared in the incoördination. Anesthesia is mentioned in 2 instances, and more or less loss of control of the sphincters in 3. These symptoms are, of course, not cerebellar. It is to be observed that nystagmus is recorded in but 5 cases; apparently in none of the others as marked as in my own patient. Although it is probable that there exists a cerebellar nystagmus, the symptom is certainly produced by lesions of other regions as well; and this series shows how frequently